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With the author's compliments.

Savill on the Electrical Changes of so-called Idiopathic Myopathy.

In the *Nouvelle Iconographie de la Salpêtrière*,[1] Dr. T. D. Savill publishes full particulars of an interesting case which corresponds in its history and clinical features with the Infantile Idiopathic Amyotrophy of Duchenne, but which, nevertheless, presents certain interesting changes in the electrical reactions of the muscles both to faradism and galvanism. Examples of this disease have generally been described as unattended by any electrical changes, and have consequently been regarded as due to inherent alteration in the muscle itself (a primitive myopathy), independent of any neuro-spinal lesion. The observation before us goes to show that this is not invariably so.

Cases of this class tend to gravitate, after visiting the different hospitals, to the metropolitan infirmaries, and Dr. Savill expresses the belief, founded on his experience at the Paddington Infirmary, that cases of so-called idiopathic or primary myopathy nearly always, at some time in their history, present electrical changes of some kind. This is contrary to the hitherto accepted view that this feature of unaltered electrical reactions is (after the age at which the symptoms first appear) the chief one which distinguishes cases of idiopathic myopathy from the larger group of progressive amyotrophic paralyses (anterior polio-myelitis). Dr. Savill further holds the view that many of the phenomena attending those cases are explicable on the assumption that there is a chronic generalised neuritis of very insidious onset and slow march. Certainly the case he describes presented, at one time, tenderness and pain along the course of the nerves and electrical changes, which would tally with this opinion. A case which tends to support this view has also been recorded by Guinon in the *Nouvelle Iconographie*,[2]

[1] May, 1894

[2] January, 1893—a case under the care of M. Brissaud in 1890.

and the number of the journal which contains the one under consideration mentions others. Brissaud also publishes several cases having the same bearing,[1] and maintains that the clinical distinction hitherto drawn between Primitive Muscular Dystrophies and the Amyotrophies of spinal origin are not borne out by the facts. Dr. Savill also points out that cases of "primitive myopathy" and of amyotrophy accompanied by the usual electrical changes occur in different members of the same family,[2] that cases of pseudo-hypertrophic paralysis, which undoubtedly belong to the class of so-called myopathies, are sometimes attended by marked electrical changes;[3] and that these circumstances all tend to support the idea that the lesion of the myopathies is, like the amyotrophies of spinal origin, primarily in the nervous system; though on account of its slight and, may be, evanescent character, it has hitherto escaped detection.

The patient was a girl named Susan S., admitted, on November 6, 1889, into the Paddington Infirmary, where she remained under observation for five years, and where she still drags on a weary existence.

The family history on the mother's side was healthy, but on the father's there was a history of epileptic and other fits, paralysis, and a tendency to drunkenness. An elder sister of the patient had certainly, from the clear account given by the mother, been attacked with identically the same form of amyotrophy, and another sister had shown some suspicious symptoms of it.

The patient, according to the mother's account, had never, even as a baby, been able to shut her eyes properly, but as a child she was bright and intelligent, and nothing abnormal was noticed about the muscles.

At the age of 7 she showed weakness and curving forwards of the spine, for which she was treated by lying upon the back, and about the same time it was noticed that the face was becoming expressionless, the speech altered, and that the lower lip was becoming very protuberant.

When about 8 years of age the mother was attracted by the commencing deformity of the feet. At the age of 9 the de-

[1] "Leçons sur les Maladiè Nerveuses," Paris, 1895, p. 344, &c.

[2] See also Charcot, "Lectures on Diseases of Nervous System," New. Syd. Soc., vol. iii., p. 64, and Cenas and Douillet, *Loire Médicale*, Nos. 7 and 8, 1885.

[3] Bédard et Rémond, *Arch. Gen. de Med.*, July, 1891.

formity and weakness of the legs was sufficient to prevent her from walking, and an operation was suggested but refused. She was never able to walk after this.

At the age of 10, though the feet were deformed, there was no general wasting obvious to the mother, and the patient was a fair height for her age. However, the weakness of the legs and face was becoming progressively worse. The involvement of the arms was so gradual that it is unknown when the deformity and wasting of these first began. It seems probable, therefore, that the atrophy started in the face as a baby, and, about the age of 7 or 8, attacked the back and legs.

Condition on Admission to the Paddington Infirmary in November, 1889, *aged* 18.—The first of the pictures shows the atrophy of arms and legs, the dropped hands, and the talipes equinus.

Figs. 2 and 3 show the same deformities, and the marked spinal lordosis in other attitudes. Fig. 2 shows very distinctly in the profile the protruding lips, which the patient was quite incapable of closing. The difficulty of supporting her accounts for the blurring of the outline of fig. 3. They need no further description. At this time there was some hyperæsthesia of the skin and some general tenderness of muscles and of the distal segments of nerve trunks.

Fig. 4 shows the maximum amount of closure of the labial and visual orifices. There was at this time an extreme degree of amyotrophy, which is described in considerable detail (*loc. cit.*).

One of the most noticeable features of this case, like others of the same kind, was the marked predominance of the atrophy of the extensors over the atrophy of the flexors, resulting in "dropped hand" and "dropped foot." These deformities were due to the tonic contraction of the remaining healthy fibres of the flexor muscles, for they could be straightened almost completely by passive movement. The power of flexing the extremities was retained to some extent, but the power of extension was almost *nil*. The extremities were always very cold and livid.

Electrical examinations, both by Dr. Savill and Dr. Kilner, showed a general *diminution of faradic contractility*. Most of the muscles required three, four, or even five times the normal strength of current to produce contraction. A notable exception to this was found in the flexors of the forearm and the flexors of the lower leg on both sides of the body, which give a reaction with about a

fourth of the normal strength of current, showing thus an increased faradic irritability.

The *galvanic contractility* was also, in general, somewhat diminished, the muscles requiring two or three times the normal strength of current to produce effect. Here again a notable exception was found in the flexors of the forearm on both sides, and the flexors of the left lower leg, where the contractility was somewhat increased, and KCC approximately equalled ACC. The galvanic contractility of the flexors of the right lower leg were normal, both quantitatively and qualitatively. The galvanic contractility was also, in general, *qualitatively* altered, so that KCC and ACC were too nearly alike, the one being barely double the other instead of $3\frac{1}{2}$ times—the only exception being the flexors of the right lower leg, as just mentioned.

Age 19.—When the patient was admitted, at the age of 18, there were some general tenderness of the muscles and nerve trunks, and some hyperæsthesia of the skin; but about the age of 19 she developed, in addition, attacks of severe pain, accompanied by acute tenderness along the nerve trunks. These symptoms became less the following year, but never entirely disappeared. Fibrillar tremors, such as Duchenne describes in these cases, were not observed in this patient, but the limbs were affected with constant fidgety movements, and an inability to keep in any one position, resembling athetosis—another symptom which might be regarded as pointing to an involvement of the nervous system.

Age 23.—But little change had taken place in the patient during the five years she had been under observation. She kept to her bed, the deformities seemed to be gradually getting more pronounced, and the weakness greater. The muscles were again examined by Drs. Kilner and Savill. There was, as before, a marked general diminution of *faradic contractility*, with the exception of the flexors of the forearm on both sides, where there was a slight increase, and excepting also the flexor muscles of the left lower leg, where the faradic contractility was approximately normal.

As regards *galvanic contractility*, there was a greater genral diminution than on the previous occasion, with the same exceptions as before, namely, the flexors of both forearms, and now *both* lower legs, where the contractility was fairly normal as to

Fig. 1.

Fig. 2.

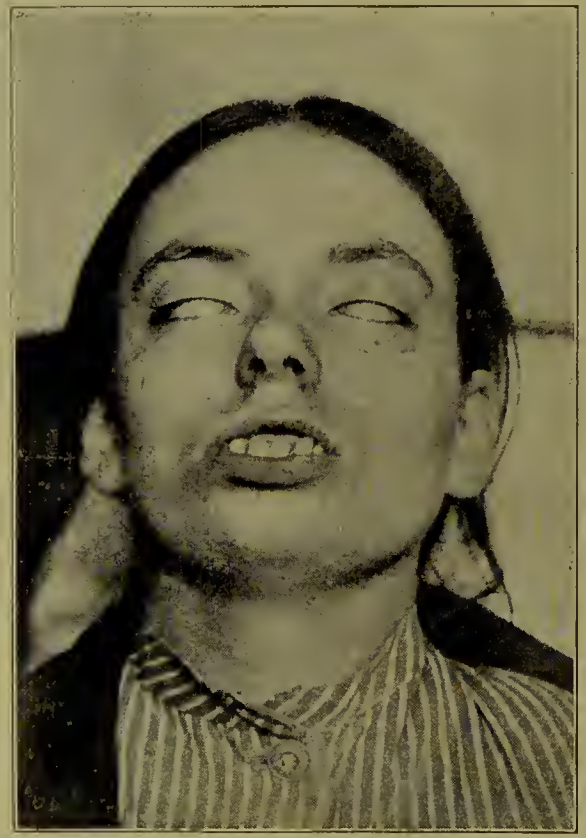

Fig. 4.

Fig. 3.

quantity; but KCC and ACC were now equal. Over the rest of the body KCC was only a little greater than ACC. These results are embodied in two tables which accompany the paper.

In general terms, the degree of alteration of the electrical reactions was proportionate to the loss of muscular substance and power. In those muscles, however, which retained some degree of power, there was, in addition to a diminution to both currents, a qualitative alteration of the galvanic contractility. On the occasion of the second examination, the muscles which retained most power were the flexors of the legs and arms. With these exceptions, the degree of atrophy was extreme.

A copious bibliography on the subject of amyotrophy is given at the end of Dr. Savill's paper. A classification of the amyotrophies is also given.

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A CLASSIFICATION OF REFLEX ACTIONS.

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THE accompanying classification is an attempt to exhibit in one view a scheme of the various groups of the reflex actions of the animal body arranged in ascending degrees of psychological complexity (*vide* column B.)

Both physiological and pathological reflexes are noted, though it was possible to quote only one or two examples of each subgroup out of a very large number.

The scheme aims at providing a more extensive and at the same time more definite terminology than is at present in use, whereby it would be possible to refer, with considerable precision, any given reflex to its appropriate genus or species.

The classification is more an indication of the lines on which such could be carried out than any final or rigid presentation of the question. It has however, I think, a certain naturalness and symmetry in its construction.

A terminology that simultaneously recognises the psychological and physiological aspects of the same action has its advantages.

We might include under "excito-motor," all those obviously simple or "low" reflexes which (*a*) do not require consciousness even for their existence ; (*b*) do not affect it (*i.e.*, the majority of them are outside its "pale") ; and (*c*) are not able to be voluntarily controlled (or "inhibited").

They are, for the most part, the actions, movements and processes of "vegetative" or "organic" life.

If, however, such a reflex affect consciousness, or be in any measure, voluntarily controlled, then it rises *psychologically* into a higher category, while its physiological nature has undergone no change.

Thus euperistalsis, conforming to *a*, *b* and *c*, is merely excito-motor ; but when consciousness becomes affected as